Photoperiodic Fertility Phenomenon (PFP): A New Hypothesis for Seasonal Respiratory Illness

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Preface

Imagine you grew up in Gabon which lies in an equatorial region of Africa. As such you have never seen trees change colors during autumn or turn dormant in the winter. In fact, you are entirely ignorant to the concept of "seasons" as you have only experienced consistent weather patterns all year long by virtue of living near the equator. For the sake of argument let's also say you have never read about different seasons or seen pictures of trees changing color in the autumn. In other words, you are completely unaware of the phenomenon of photoperiodism - the physiological response of organisms to the relative lengths of light and dark periods during different seasons. On the other hand, as a medical physician, you are very familiar with the concept of "germ theory" and the idea that disease is caused by microscopic pathogens.

Now, let's say you take a trip to Vermont in the United States, which is well known for its foliage during autumn. Apart from the chilly weather, you experience for the first time in your life the phenomenon of trees exhibiting bright foliage only to drop their leaves and seemingly die en masse. You can hardly believe your eyes as over the course of a few weeks so many trees fall victim to some sort of plague. As a physician well versed in germ theory, you are tempted to say there is a contagion which targets the leaves of trees and spreads through the rest of the tree, perhaps through the tree's circulatory system. While you are not a virologist, you believe the contagion at play must be a virus as you can observe many healthy trees acquire the same symptoms and inevitably die. Though you can not see the virus or properly isolate it through experimentation you assume it must be a virus since no bacteria can be found in the samples you collect from trees. In addition the presumed virus only targets trees since no people or animals exhibit signs of infection. Finally, it appears the virus targets special cell receptors in the leaves of deciduous trees because a few trees, the coniferous ones that lack broad leaves, seem to be protected from the contagion. Something about the immune system of pine trees protects them and you think their needles or pine cones must produce antibodies to the presumed virus.

You contact colleagues in Gabon and notify them of the rapidly spreading virus and that you are worried your agricultural industry back home could be at risk. In response to your report, reconnaissance is sent to other parts of the world to investigate the possible spread of the virus. Sure enough trees in Canada, Sweden, Japan, Italy, and many other countries are all showing signs of the contagion. Fortunately it appears the virus has not transferred to equatorial regions of the world but in order to prevent the spread, Gabon implements a trade embargo on any agricultural imports from northern countries. This cripples your economy but you can not risk inadvertently importing a virus that could decimate your country's crop production. As the first to discover the contagion (which is presumed to be a virus) you are given multiple grants to develop chemicals to protect trees and crops. You come across a specific chemical compound

that appears to prevent some trees from dropping all their leaves. While trees that are sprayed with the chemical still exhibit signs of the virus they do not drop all their leaves so you believe your treatment is working to a reasonable extent (unbeknownst to you these trees are just oak and beech trees which don't drop their dead leaves in the winter). Gabon's main environmental agency in collaboration with private companies develops multiple variations of your chemical treatment and applies it to trees and other crops in your home country in order to prevent a viral outbreak. In rare instances some trees and crops exhibit bad reactions to the treatment and even die but that is a small price to pay to be sure the presumed virus does not decimate your entire crop production networks. This entire ordeal has taken place over about five months and then a funny thing happens. All the trees you assumed to be dead from the presumed virus begin to bloom around April and you are informed that the bizarre phenomenon you witnessed actually takes place every year as a product of photoperiodism - the seasonal change in daylight hours. You are told there is nothing to worry about and the trees in Vermont do this every year and the reason you have never seen it before is because trees around the equator do not have any photoperiod response. Do you admit to your colleagues back in Gabon you were wrong and there was never any contagion or virus?

Figure 1. Foliage in Colorado. If you didn't know any better (i.e. if you use germ theory to explain everything) you might think the yellow trees in this image have contracted some sort of virus that makes them change color and drop their leaves. You might also think the pine trees are immune to said virus as they retain their green color and don't drop their needles. Of course there is no virus at play and the reality is deciduous trees go through an annual process of changing color and dropping their leaves as a product of photoperiodism - the seasonal change in daylight hours. PFP suggests human beings go through an analogous photoperiod response every winter that manifests as seasonal illness like the common cold, the flu and COVID-19.

Abstract

Photoperiodism is a biological mechanism by which organisms respond to changes in day length and has been shown to play an important role in regulating seasonal patterns of fertility in a variety of species. In humans, changes in photoperiod are thought to influence the production of hormones such as testosterone, luteinizing hormone and follicle-stimulating hormone, which play key roles in regulating sperm production, ovulation and fertility. Despite the correlation between photoperiodism, human fertility and seasonal illness, the theoretical connection between all of them has not been made and the precise mechanisms underlying the effects of photoperiodism on human fertility and health remain poorly understood. In this paper, I provide a general overview of how photoperiodism regulates fertility in a variety of species and posit a new hypothesis on photoperiodism, fertility and seasonal illness in humans which I call Photoperiodic Fertility Phenomenon (PFP). PFP is an evolutionary adaptive trait in which humans have a biological response to shortened day length and temporarily become more fertile. The temporary increase in hormone levels during this time window (usually coinciding with low photoperiod in winter) is the actual cause of perceived seasonal illness as opposed to viruses or some contagion. PFP assumes illnesses like the common cold, influenza, and COVID-19 are natural physiological responses to certain photoperiods and should not actually be considered illnesses or contagious diseases. Ultimately, a better understanding of the role of photoperiodism in human reproduction and health may have important implications for the development of interventions aimed at improving fertility and ending the medical industrial complex's obsession with vaccines to treat perceived seasonal illness.

Examples of Photoperiodism

Photoperiodism in plants

Photoperiodism is the physiological response of plants and animals to changes in day length or photoperiod. The phenomenon was first discovered in the early 20th century by scientists who noticed that certain plants only flowered under specific day lengths [1,2]. Over the years, researchers have made significant strides in understanding how photoperiodism works and the underlying mechanisms involved. One of the earliest and most notable examples of photoperiodism was observed in the tobacco plant, Nicotiana tabacum. In 1912, the American botanist Garner and his British colleague Allard noticed that tobacco plants only flowered when exposed to a certain amount of light per day [3]. They found that the plants required a critical photoperiod of 14 hours of light per day to initiate flowering, and that any deviation from this photoperiod would delay or prevent flowering altogether. This discovery led to a new field of research known as photoperiodism, which is now a major area of study in plant physiology. Since then, many other plant species have been found to exhibit photoperiodism, including rice, wheat, soybean, and many others*. Generally speaking, researchers determined plants fall into three distinct groups based on their photoperiodic responses: short-day plants, long-day plants, and day-neutral plants [4]. Short-day plants, like chrysanthemum, rice, soybean, onion, are plants that flower during days which are shorter than a critical day length. These plants bloom when daylight hours drop below a particular critical threshold, typically during the late summer

or autumn. Long-day plants like coneflower, lettuce, spinach, are plants that flower during days which exceed a critical day length. These plants bloom in summer when daylight hours exceed a certain threshold. Plants which flower regardless of day length are considered day-neutral. Some day neutral plants include cucumber, corn, tomato as the flowering process is not regulated by the length of daylight hours and is instead regulated by the maturity of the plant. One important aspect of photoperiodism is its effect on plant germination. In many plant species, the timing of seed germination is regulated by the photoperiod, and this can have significant effects on the growth and survival of the plant. For example, some plants require a specific photoperiod to initiate germination. If the seeds are planted during a time of year when the day length does not match their photoperiod requirement, they may not germinate at all, or they may germinate poorly. This can be a disadvantageous trait for a plant species, as it may limit their ability to establish themselves in new habitats. On the other hand, in some plant species, the timing of seed germination is regulated by the photoperiod as a way of ensuring that the seedlings emerge during the optimal time of year for growth and survival. For example, if a plant species requires a long photoperiod for germination, it may ensure that its seeds only germinate during the long days of summer, when there is ample sunlight and warmth to support growth. This can be an advantageous trait for a plant species, as it increases the likelihood of successful seedling establishment and survival. The evolution of photoperiodism in plants is thought to be driven by natural selection, with plants evolving to match their photoperiodic requirements to the environmental conditions in which they live. For example, plants growing at high latitudes, where the day length varies dramatically over the course of a year, have evolved to exhibit photoperiodism as a way of synchronizing their growth and reproduction with the changing seasons [5]. In this way, photoperiodism is an adaptive trait that allows plants to survive and reproduce in a wide range of environmental conditions. In summary, photoperiodism can have significant effects on plant germination, and it can be an evolutionarily adaptive trait. By regulating the timing of seed germination in response to changes in day length, plants are able to increase their chances of successful establishment and survival in different environmental conditions.

*More examples of photoperiodism in plants include: the poinsettia, which is a popular holiday plant that is known for its bright red and green leaves. Poinsettias require long nights to flower, and the plant is often used as a model system for studying the mechanisms of photoperiodism. Scientists have found that the plant's flowering is controlled by a group of genes that respond to changes in day length, which in turn regulate the production of a hormone called florigen that stimulates flowering. Another example of photoperiodism in plants is the potato. The potato is a member of the Solanaceae family, which also includes tomatoes, peppers, and eggplants. Potato plants require short days to initiate tuberization, which is the process of forming underground storage structures that are used to store nutrients over the winter. Researchers have found that this response is regulated by a complex network of genes that are sensitive to changes in daylength and temperature. In addition to flowering and tuberization, photoperiodism also plays a role in many other physiological processes in plants, such as seed germination, stem elongation, and leaf growth. Understanding the mechanisms of photoperiodism is therefore critical for improving crop yields and developing new varieties of plants that can grow and thrive under different environmental conditions.

Photoperiodism in insects

Photoperiodism is not limited to plants and is also observed in insects. Insects use photoperiodism to regulate a variety of physiological and behavioral processes, including diapause, migration, and reproduction [6]. Diapause is a form of dormancy observed in many insects, and it is triggered by changes in photoperiod. Insects that enter diapause are able to survive unfavorable conditions, such as cold temperatures or lack of food, by slowing down their metabolism and conserving energy. The timing and duration of diapause is regulated by photoperiod in many insect species. For example, some species of mosquitoes enter diapause in response to shortening day lengths, which occurs in the autumn when temperatures begin to cool. Photoperiodism also plays an important role in the timing of insect migration. Many species of butterflies and moths migrate seasonally to take advantage of the changing availability of food and resources. The timing of migration is regulated by photoperiod, with insects typically beginning their migration when the days begin to shorten and temperatures cool.

Photoperiodism can affect insect reproduction in a variety of ways, and the specific effects vary depending on the insect species [7]. Some examples of how photoperiodism affects insect reproduction include: Timing of mating: Many insect species use photoperiodism to regulate the timing of mating. For example, female mosquitoes require a specific photoperiod to initiate mating behavior, and males often use the length of the day to determine when to begin searching for mates. In some species, such as the cabbage looper moth, photoperiod can also affect mate choice and sexual selection. Egg development: Photoperiodism can also affect the timing of egg development in insects. For example, in some species of fireflies, females require a specific photoperiod to develop their eggs, with longer day lengths resulting in larger and more numerous eggs. Diapause termination: Insects that enter diapause, a form of dormancy, can use photoperiod as a cue to terminate diapause and begin reproductive activity. For example, in the Colorado potato beetle, photoperiod can trigger the termination of diapause and the onset of reproductive behavior. Offspring development: The photoperiod experienced by a female insect can also affect the development of her offspring. For example, in the bean beetle, a longer photoperiod during the pre-oviposition period can result in larger and more fecund offspring. Reproductive diapause: Some insect species enter a reproductive diapause, where they are able to mate and produce eggs, but the eggs do not develop until a later time. Photoperiod can regulate the timing of reproductive diapause, with some species requiring a specific day length to initiate or terminate the diapause. In summary, photoperiodism is a phenomenon observed in many insects, and it plays an important role in regulating a variety of physiological and behavioral processes, including diapause, migration, and reproduction. By responding to changes in day length, insects are able to synchronize their behavior and physiology with the changing seasons, which can increase their chances of survival and reproductive success.

Photoperiodism in birds

Photoperiodism plays an important role in regulating the life cycle of many bird species, including the timing of breeding, migration, and molting [8]. Some examples of how photoperiodism affects birds include: Timing of breeding: Many bird species use photoperiod as

a cue to initiate breeding behavior. As the days lengthen in the spring, birds may become more active and territorial, and begin to mate and build nests. The specific photoperiod required to trigger breeding can vary depending on the species, with some requiring longer or shorter day lengths than others. Migration: Photoperiod can also play a role in regulating bird migration. For example, some bird species use changes in day length to determine when to begin their autumn migration, while others may use photoperiod to time their arrival and departure from breeding grounds. Molting: While photoperiod can influence the timing of molt in some bird species, it is not the sole cause of molting. Molting is a complex process that involves changes in hormones and physiology, as well as environmental cues such as temperature and food availability. That being said, photoperiod can play a role in the initiation of molt, with some species beginning to molt as day lengths shorten in the autumn. Song production: Photoperiod can also influence the timing and intensity of song production in many bird species. For example, male songbirds may begin singing more frequently and loudly as the days lengthen in the spring, as they establish territories and attract mates.

Photoperiodism can affect the reproductive organs of birds by regulating the secretion of hormones that control reproductive activity. Specifically, changes in day length can stimulate or inhibit the release of gonadotropin-releasing hormone (GnRH) from the hypothalamus of the brain. GnRH then stimulates the pituitary gland to release follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which stimulate the development of the reproductive organs and the production of sex hormones like estrogen and testosterone. In male birds, changes in day length can affect the development of the testes and the production of sperm. For example, in some bird species, increasing day length in the spring stimulates the growth of the testes and the production of high-quality sperm. In contrast, decreasing day length in the autumn can cause the testes to regress and sperm production to decrease. In female birds, photoperiodism can regulate the development of the ovaries and the production of eggs. For example, in some bird species, increasing day length in the spring stimulates the development of the ovary and the production of high-quality eggs. In contrast, decreasing day length in the autumn can cause the ovary to regress and egg production to decrease. Overall, photoperiodism plays an important role in regulating reproductive activity in birds, and the specific responses of different bird species can vary depending on a variety of factors, including their evolutionary history and ecological context.

Figure 2. You might think the cardinal on the left has contracted some sort of flesh eating bacteria or virus (especially if you are a virologist) but in reality it's just going through a photoperiod response to fewer daylight hours in autumn. The molting process is evolutionarily adaptive as the male cardinal loses his brilliant red feathers in the autumn thereby making him less likely to attract a mate and have offspring right before and during winter, which would be the WORST time to reproduce. By the end of winter his brilliant red feathers will have returned and the mating game is back on just in time for spring, the most evolutionary adaptive time window for cardinals to reproduce.

Photoperiodism in mammals

In mammals, the photoperiodic system is controlled by a specialized area of the brain known as the suprachiasmatic nucleus (SCN), which is located in the hypothalamus [9]. The SCN receives input from the eyes via the optic nerve and is responsible for synchronizing circadian rhythms in the body with the external environment. When the SCN detects changes in day length, it sends signals to the pineal gland, a small gland located in the brain, to regulate the secretion of the hormone melatonin. Melatonin is involved in the regulation of many physiological processes, including the sleep-wake cycle and seasonal behaviors [10]. In mammals, changes in melatonin secretion can trigger reproductive activity, such as the onset of puberty and breeding season, and can also regulate hibernation and migration. For example, in some mammals, the shortening of daylight hours in the autumn triggers an increase in melatonin secretion, which in turn stimulates the onset of winter hibernation or migration. In other species of mammals, increasing daylight hours in the spring triggers a decrease in melatonin secretion, which stimulates the onset of the breeding season. The specific photoperiodic responses of different mammal species can vary depending on a variety of factors, including their evolutionary history, ecological context, and genetic makeup. Therefore, understanding the photoperiodic responses of different mammal species can provide valuable insights into the regulation of seasonal behaviors in the animal kingdom.

Overall photoperiodism can affect reproduction in mammals by regulating the secretion of hormones that control reproductive activity [11]. A few examples include: Seasonal breeding: Many mammals have a seasonal breeding pattern, which is regulated by changes in day length. For example, in sheep, decreasing day length in the autumn stimulates the secretion of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the pituitary gland, which stimulates the development of ovarian follicles and the production of estrogen. This triggers the onset of the breeding season. Conversely, increasing day length in the spring inhibits the

secretion of FSH and LH, which causes the ovaries to regress and the breeding season to end. It should be noted that mammals can be both short day breeders (like sheep that breed in the autumn) and long day breeders (like horses which breed in the summer). Reproductive suppression: In some mammals, changes in day length can also trigger reproductive suppression. For example, in female mice, exposure to short days (less than 12 hours of light per day) inhibits the secretion of FSH and LH, which prevents the development of ovarian follicles and suppresses the production of estrogen. This can delay or even prevent the onset of the breeding season. Overall, photoperiodism plays an important role in regulating reproductive activity in mammals, and the specific responses of different mammal species can vary depending on a variety of factors, including their evolutionary history and ecological context.

The effect of latitude on photoperiodism

Latitude can affect photoperiodism in plants and animals due to variations in day length at different latitudes. As the seasons change, the amount of daylight and darkness varies depending on location. At the equator, day length is fairly consistent throughout the year, with around 12 hours of daylight and 12 hours of darkness. However, as one moves closer to the poles, the length of daylight and darkness changes more dramatically throughout the year. In the summer months, regions near the poles experience long days with little darkness, while in the winter months, they experience long nights with little daylight. This variation in day length can trigger the photoperiodic response in plants and animals, which can have important effects on growth, development, and reproduction. Generally speaking, the higher latitude one travels the more species exhibiting photoperiodism one will find. This is because species have evolved different photoperiodic responses to adapt to their local environments.

Hypothesis

Typical seasonal illnesses like the common cold, influenza, and COVID-19 are not an immune response to viral infection and are instead an endocrine response to seasonal changes in daylight hours in northern and southern hemispheres respectively. The shortening of daylight hours in conjunction with the lengthening of night time hours triggers endocrine changes with evolutionary adaptive effects and should be considered part of human reproductive strategy. More specifically, hormone changes during short day periods (i.e. autumn or winter) increase fertility and the likelihood of conception and briefly manifest as a seasonal respiratory illness in the following weeks. The timing of the endocrine changes and conception is evolutionary adaptive in that gestation coincides with summer allowing for maximum food acquisition for the pregnant mother. Likewise, birth of offspring is more likely to occur in late summer or autumn when food stockpiles are most abundant. While not fully elucidated in this paper the exact biological mechanism is most likely mediated by the pineal gland in relation to the hypothalamus and humans of any age and sex can experience the phenomenon even if they are not of reproductive age (i.e. everyone can experience a seasonal illness).

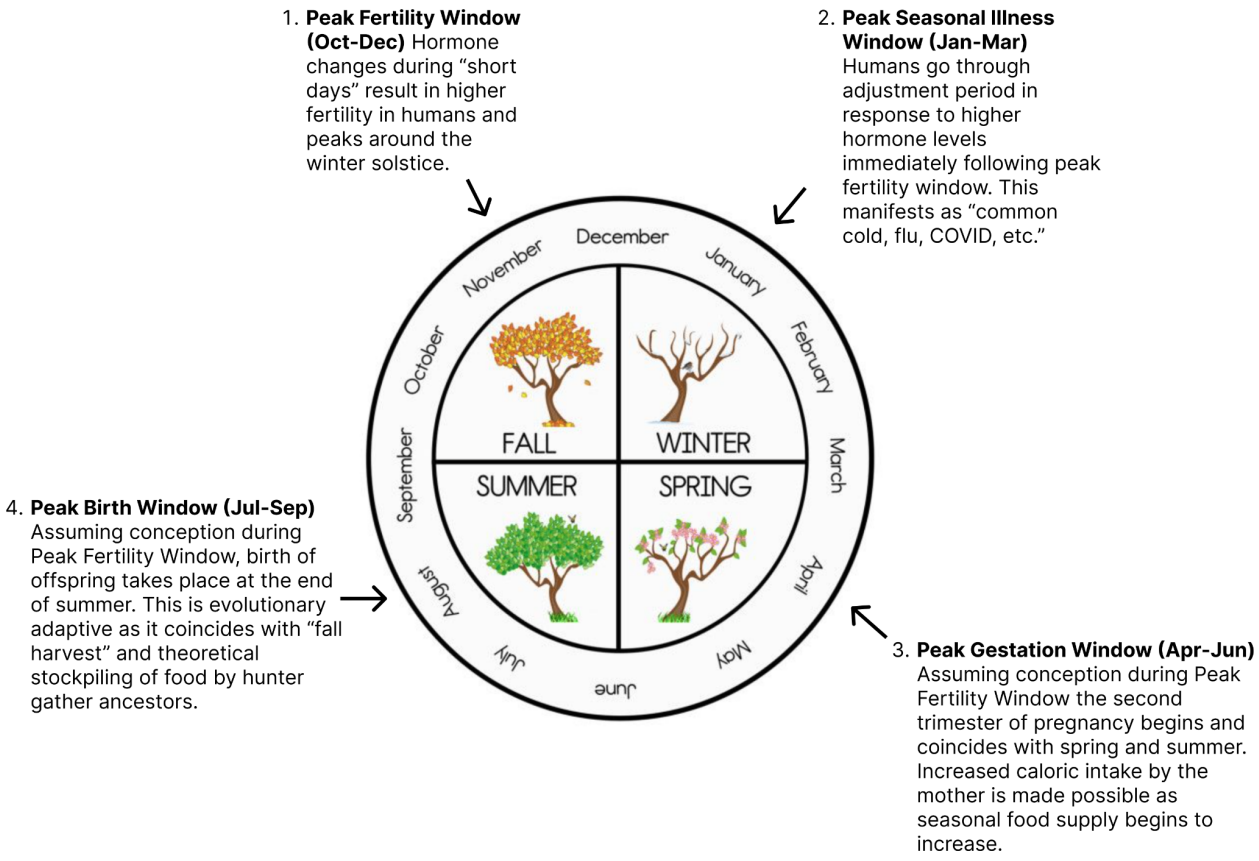
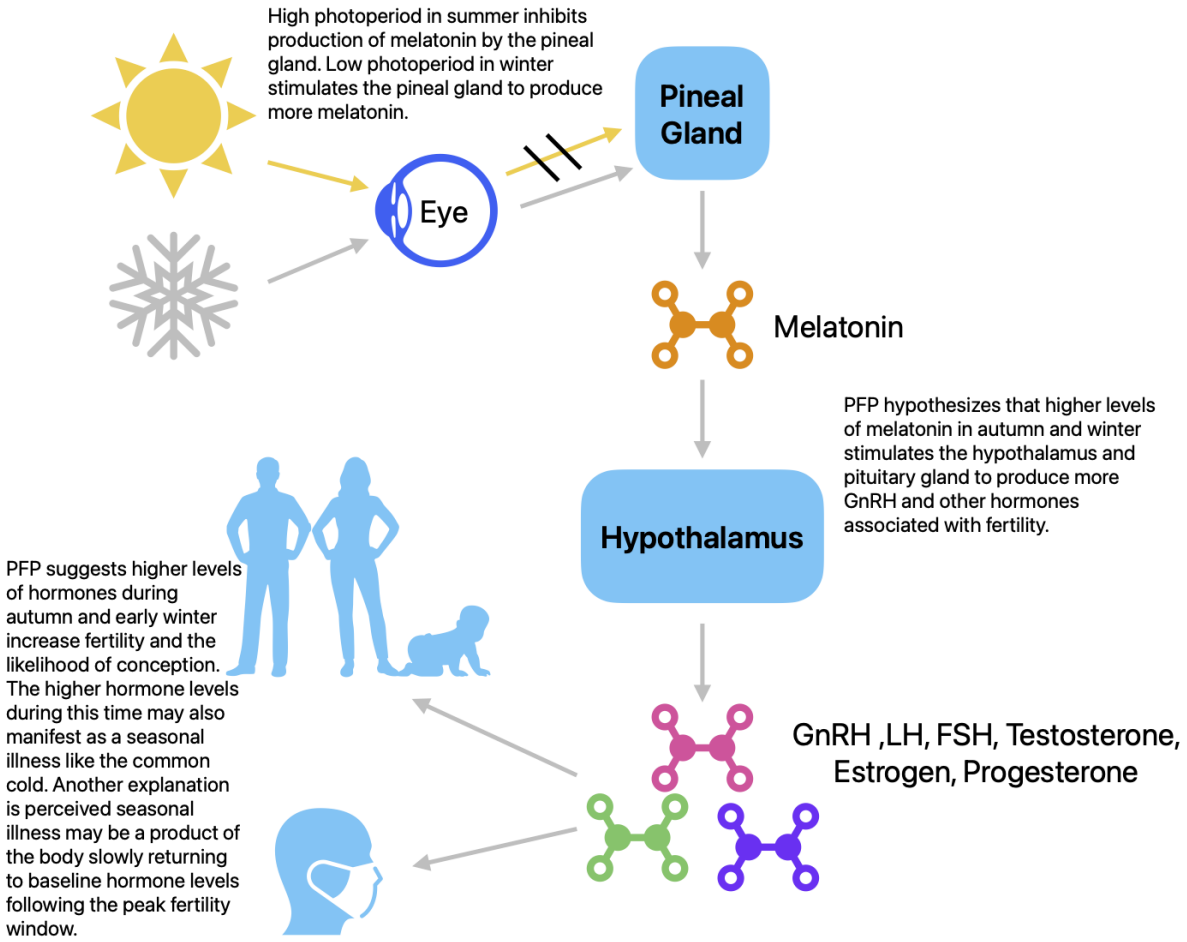


Figure 3. Photoperiodic Fertility Phenomenon (PFP) in the northern hemisphere. The annual phenomenon is "reversed" in the southern hemisphere, with peak fertility window and seasonal illness occurring around June-July and peak birth window occurring in March. In the modern world with grocery stores and fast food we take access to food for granted. Yet for our hunter gatherer ancestors access to food was highly dependent on the seasons with most fruits and vegetable harvests taking place in late summer and autumn. Likewise big game like deer and elk was probably most abundant in autumn. PFP assumes human biology has been hardwired so that conception is most likely to occur in late autumn or early winter so that birth occurs in late summer when food is most abundant for mother and child. In reality the hormone/fertility changes in late autumn or early winter are the real cause of perceived seasonal illness as opposed to the prevailing notion that viruses or contagion is the cause.

Evaluation of Hypothesis

Photoperiodism as an evolutionary adaptive trait

As the saying goes "everything happens for a reason" and in any case, weather it be plants, insects, birds or mammals, photoperiodism should be considered evolutionarily adaptive. Photoperiodism allows organisms to time their life cycle events (e.g., reproduction, migration, hibernation) with seasonal changes in their environment. By doing so, organisms can maximize their chances of survival and reproductive success. Considering this, perceived seasonal illness in humans may not be a product of some random viral mutation or any contagion for that matter but rather a hormonal change during shorter photoperiods in autumn and winter. It is well understood that photoperiod affects the pineal gland which in turn changes the level of melatonin in human beings. Moreover, it is well understood that melatonin can affect hormone levels in mammals and alter fertility. PFP asserts the fluctuations in hormones in late autumn and winter can briefly cause perceived seasonal illness in the following weeks. These perceived seasonal illnesses may be a sort of "withdrawal" period after the peak fertility window during which the body will adapt to and recover.



Seasonal patterns of fertility in humans - evidence of PFP?

Seasonal patterns of fertility in humans have been observed in various parts of the world, with peaks in conception rates occurring during certain times of the year. In general, the seasonal pattern of fertility in humans is characterized by higher conception rates around the winter solstice in populations living in the northern and southern hemispheres respectively [12]. This leads to higher birth rates nine months later in summer and early autumn. As such the most common birth month in the northern hemisphere is usually July or August and the most common birth month in the southern hemisphere is usually March or April. These trends may be evidence for PFP as changes in photoperiod during short days and may lead to increased production of hormones such as luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which play key roles in regulating ovulation and fertility in women. In addition, lower temperatures during the winter months may also contribute to increased fertility by improving sperm quality and increasing sexual activity. There are less seasonal patterns of fertility exhibited in equatorial regions and fertility patterns, if any, may have cultural explanations. Likewise there is little to no trend of seasonal illness in equatorial regions which may also be evidence of PFP.

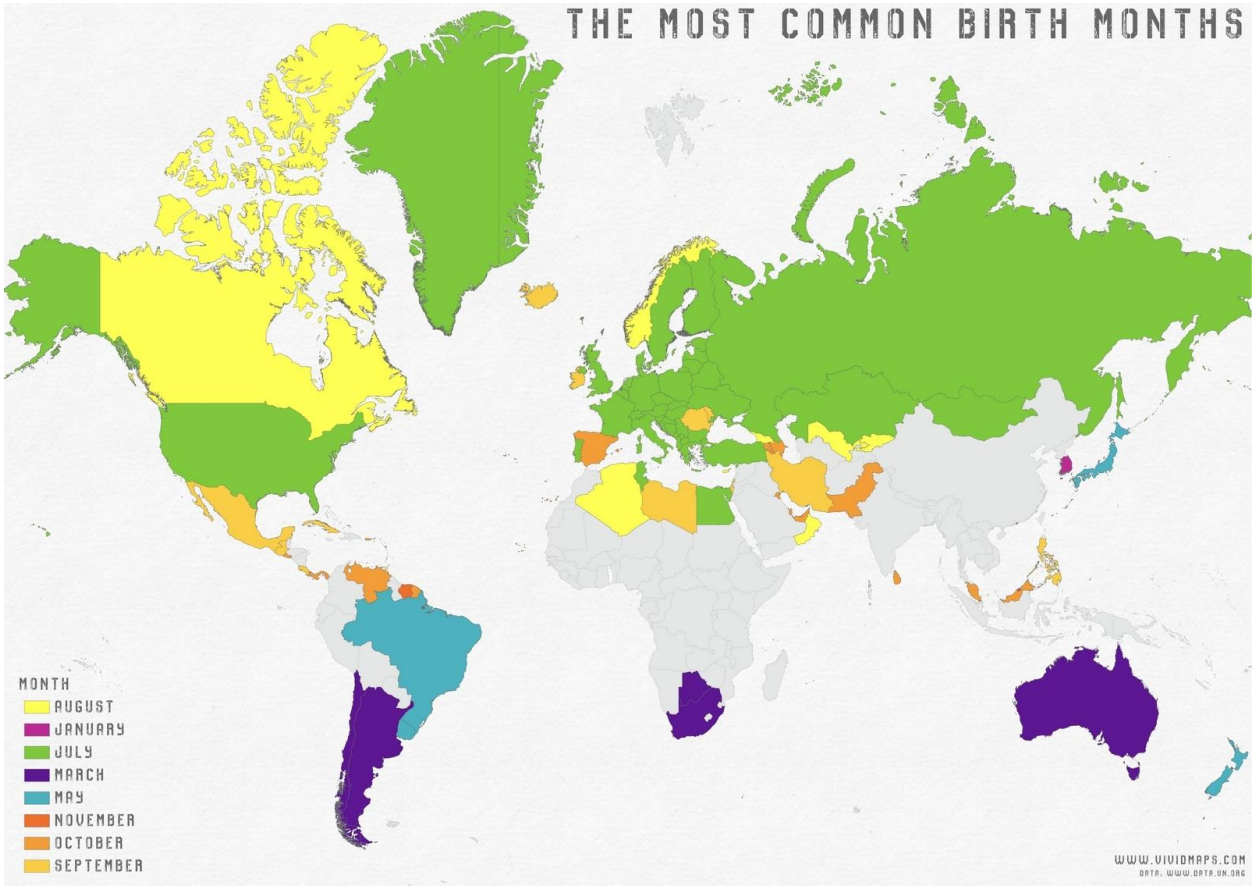


Figure 4. The difference in most common birth months in the northern and southern hemispheres is not a coincidence or cultural phenomenon. According to PFP, fertility in the northern hemisphere increases in autumn and peaks around the winter solstice thereby placing peak births in July and August. Conversely, fertility in the southern hemisphere increases during their respective autumn and winter thereby placing the most births in March.

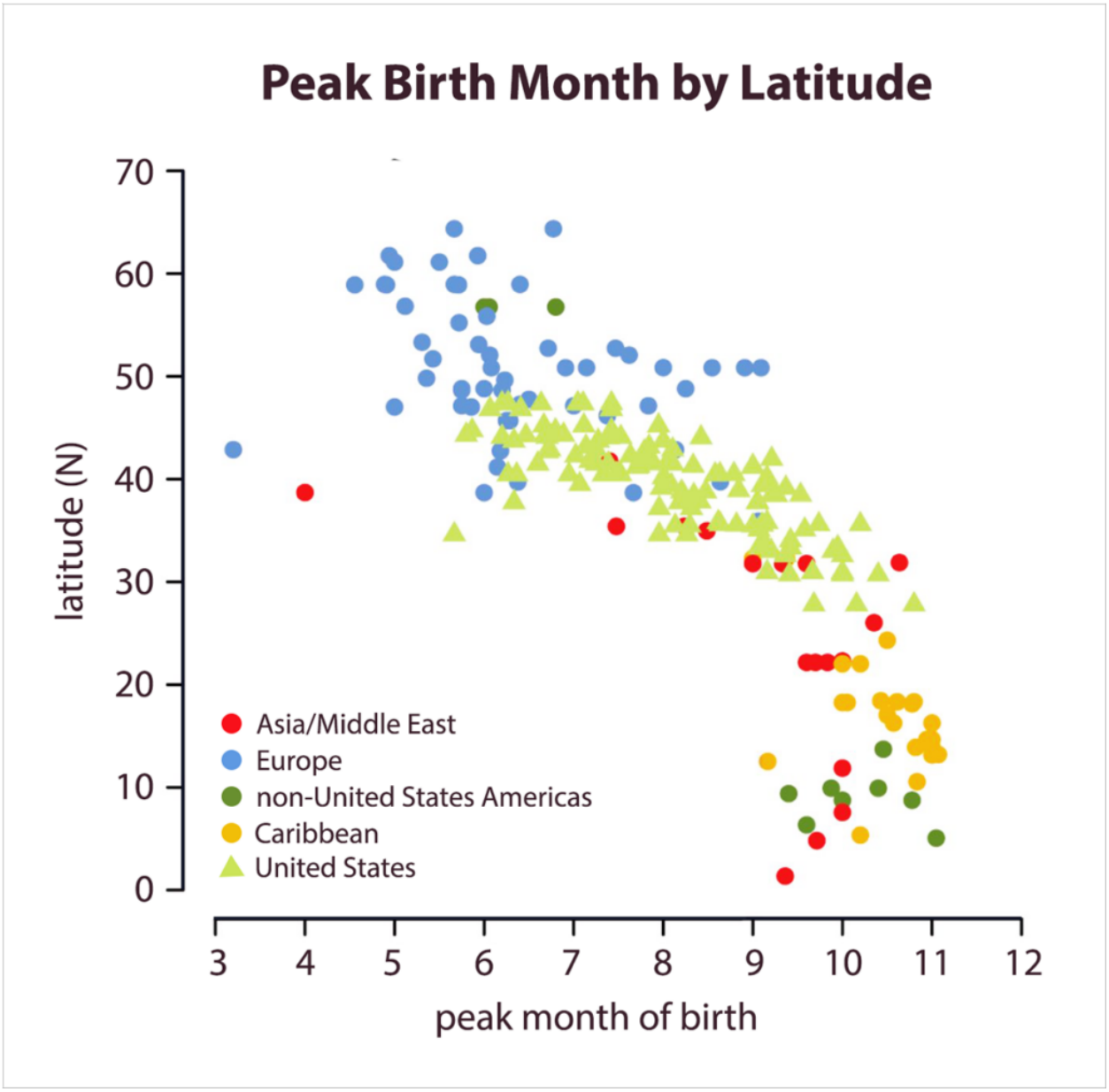


Figure 5. The correlation between latitude and birth month may provide more evidence of PFP. The further north one travels the more "forward" in the year births will occur. This suggests photoperiod affects fertility and the likelihood of conception in that the more pronounced the photoperiod change (i.e. the further north one travels and experiences darker winters) the higher effect on fertility. This makes sense from an evolutionary perspective as the further north a population lives the more important it is for them to time births to occur earlier in the summer.

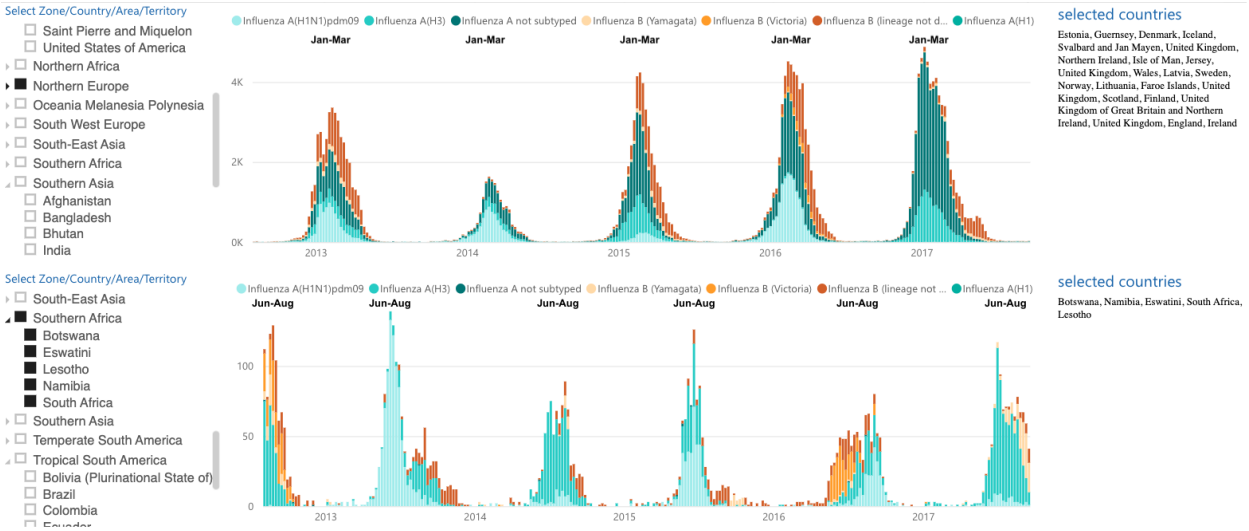


Figure 6. Most people are aware of the seasonality of illnesses like the common cold, flu and COVID-19. The prevailing wisdom suggests seasonality is explained by the fact people are more likely to stay indoors in winter and thus pass pathogens to each other and that viral particles are more capable of moving through colder and less dense air in winter. PFP suggests viruses have nothing to do with seasonal illness and instead illnesses like the flu are explained by low photoperiod during winter months. In the above image we can see how annual influenza patterns are "reversed" between northern and southern hemispheres peaking just after each respective winter solstice when photoperiod is the lowest.

Seasonal pattern of heart attacks - evidence of PFP?

There is evidence to suggest that the risk of cardiac deaths in humans follows a seasonal pattern, with higher rates occurring around the winter solstice [13]. To date, the exact mechanisms underlying this pattern are not entirely understood and while there are several previous explanations of seasonal trends in cardiovascular events they don't fully elucidate the phenomenon. For example, it's thought exposure to cold temperatures can increase blood pressure and cause constriction of blood vessels, which in turn can lead to an increased risk of heart attacks and other cardiac events [14]. The problem with this explanation is that warmer states in the southern US actually exhibit higher trends of cardiovascular deaths when compared to the colder northern states so cold temperature may not actually be the primary factor at play [15]. Some have suggested changes in physical activity levels and diet may also play a role in seasonal trends in cardiac deaths. During the winter months, many people tend to be less physically active and consume more high-calorie, high-fat foods, which can increase the risk of obesity, high blood pressure, and other risk factors for heart disease. That being said, this explanation is somewhat anecdotal and people may consume high-calorie, high-fat foods during other holidays and sporting events without experiencing higher risk of heart attacks [16] so the "Christmas cookie" may not explain the annual spike in cardiac deaths. Considering cardiovascular events in the southern hemisphere are actually lowest around Christmas the trend of heart attacks is therefore not explained by behavior correlating with Christmas and instead correlates with the winter solstice in each respective hemisphere [17]. In all likelihood, seasonal trends in cardiac deaths may be a product of photoperiod on the body's endocrine system. Changes in day length and light exposure can affect the body's production of hormones

and other factors that play a role in heart health. More specifically there may be an increase in testosterone in men around the winter solstice, as a product of PFP, which increases the buildup of cholesterol resulting in more cardiac events. Overall, while the exact mechanisms underlying seasonal trends in cardiac deaths are not fully understood, endocrine changes in response to short photoperiod may be the most likely explanation. As discussed the endocrine changes primarily serve to increase fertility around the winter solstice and the increase in cardiovascular events and perceived seasonal illness may be an unfortunate byproduct and are mainly only a risk to the elderly.

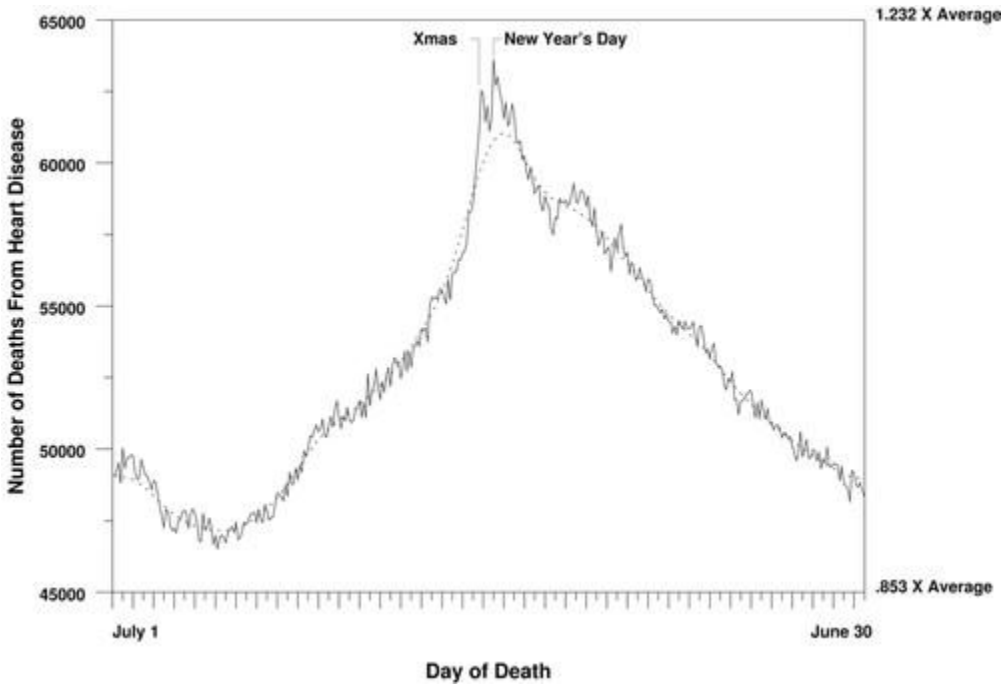


Figure 7. Daily US cardiac deaths, 1973–2001. Cardiac events appear to be correlated with Christmas and New Year's but in reality they are correlated with the winter solstice (i.e. when there are the fewest amount of daylight hours). The low photoperiod affects hormones which may affect cholesterol levels and cause cardiac deaths.

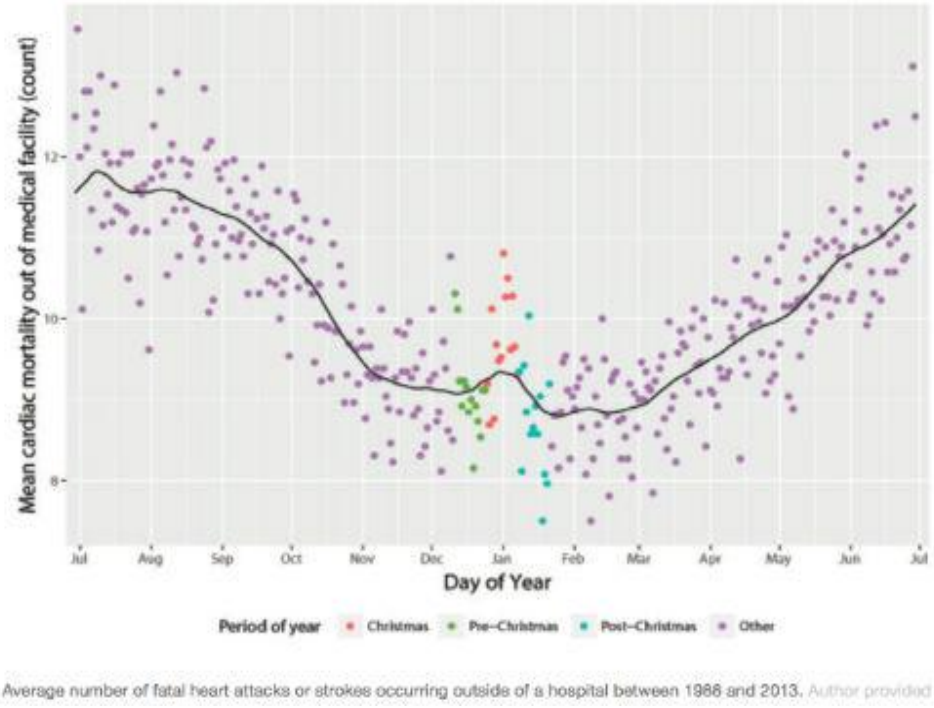


Figure 8. Cardiac deaths in Australia. The figure clearly shows more deaths during their respective winter (edges of the figure) than in summer (middle of the figure). This implies cardiac deaths are not a product of diet trends around Christmas and instead are a product of low photoperiod in winter - yet more evidence of PFP.

Menstrual synchrony, loss of taste and smell - evidence of PFP?

Menstrual synchrony, also known as the "McClintock effect," is the phenomenon in which menstrual cycles of women who live together or spend a significant amount of time together become synchronized over time [18]. This phenomenon has been observed in a number of different settings, including among female roommates, co-workers, and members of the same sports teams. The exact mechanisms underlying menstrual synchrony are not fully understood, but it may actually be more evidence for PFP which would suggest menstrual synchrony may occur under very strict conditions where women who live together and follow the exact same daily routine are thus exposed to the same daily photoperiod. In these cases, the daily and nightly levels of hormones such as melatonin, estrogen and progesterone align due to the same photoperiod resulting in the menstrual synchrony. This is contrary to current explanations, which have been difficult to reproduce under experimental conditions, which suggest pheromones or social cues explain menstrual synchrony. Experimental designs that bear in mind the theory of PFP may be able to reproduce the phenomenon of menstrual synchrony by having female subjects follow the exact daily routine with the same sunlight exposure as opposed to just living or spending time together.

When people experience a perceived seasonal illness like the common cold, they may experience a temporary loss or reduction in their sense of taste or smell. Contrary to the current theory which suggests this is caused by a viral infection, it may actually be caused by endocrine changes during low photoperiod in winter. There is substantial evidence to suggest endocrine

changes affect sense of taste and smell [19]. For example, during pregnancy, women may experience heightened sensitivity to certain odors and tastes, and they may find that foods they previously enjoyed now taste unpleasant. This is thought to be due to the increase in estrogen and progesterone levels during pregnancy, which can affect the olfactory system and the taste buds. Similarly, hormonal changes during menopause can also affect a woman's sense of smell and taste. As estrogen levels decline during menopause, some women may experience a decreased ability to taste and smell certain foods. This can be particularly pronounced for bitter and sweet tastes. Hormonal imbalances and disorders can also affect the sense of smell and taste. For example, hypothyroidism, which is a condition in which the thyroid gland doesn't produce enough hormones, can lead to a decrease in the sense of smell and taste [20]. This is thought to be due to the fact that the thyroid hormones play a role in the development and maintenance of the olfactory system. Overall, hormones can play a role in influencing the sense of smell and taste, both through changes in hormonal levels and through their effects on the development and function of the olfactory system and the taste buds. As such, the loss in sense of taste and smell during perceived illness like the common cold, flu, and COVID-19 may not be a product of viral infection and rather be more evidence for PFP.

Discussion

Many critics of virology have pointed out the inability for virologists to properly isolate and purify viruses. The Perth Group has written extensively that HIV, the alleged virus that causes AIDS, has never properly been isolated and that AIDS itself may in fact be a combination of existing diseases rebranded as a new phenomenon [21, 22]. Likewise, Kary Mullis, inventor of polymerase chain reaction (PCR) technology came to similar conclusions, leading him to suggest PCR should not be used to identify the presence of alleged viruses in human subjects [23,24]. Even molecular biologists like Peter Duesberg have pointed out flaws in the viral model of infectious disease [25,26] only to be blackballed from mainstream public health discourse. Looking further back in history we can find evidence, or rather lack thereof, regarding the germ theory of contagion. Rosenau tried to demonstrate Spanish Flu was contagious only to find none of his healthy subjects came down with the illness when they were exposed to the alleged contagion, leading him to proclaim “we entered the outbreak with a notion that we knew the cause of the disease, and were quite sure we knew how it was transmitted from person to person. Perhaps, if we have learned anything, it is that we are not quite sure what we know about the disease” [27]. Even those responsible for developing germ theory in its early days were apparently aware of its pitfalls and Pasteur himself admitted “Bernard was right; the pathogen is nothing, the terrain is everything” [28].

Considering the possibility that germ theory may not explain seasonal illness, many of its critics have been gaining support but these critics have not provided an adequate alternative explanation for seasonal illness. Time and time again, virology critics are met with the same rebuttal: “OK you have raised good points regarding the problems of virology, especially relating to isolation and purification. But if a virus is not making people sick, what is? What makes you ill?” There are clearly “illnesses” that can be observed in people occupying similar geographic areas. Moreover these “illnesses” transpire in a seasonal fashion almost exclusively during winter. There must be some explanation for the phenomenon of seasonal illness since it is not random from a time and space perspective and may not be from bacteria or viruses. For those

skeptical of virology and the theory of contagion you now have a new explanation for seasonal illness: Photoperiodic Fertility Phenomenon - seasonal changes in the human endocrine system as a product of reduced daylight hours.

Acknowledgments

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